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10/623,039	07/18/2003	Subhashis Banerjee	BBI-8188	1401

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EXAMINER	
BLANCHARD, DAVID J	

ART UNIT	PAPER NUMBER
1643	

NOTIFICATION DATE	DELIVERY MODE
07/09/2008	ELECTRONIC

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

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Office Action Summary	Application No.	Applicant(s)	
	10/623,039	BANERJEE ET AL.	
	Examiner	Art Unit	
	David J. Blanchard	1643	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *24 March 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,3,4,12,18,22,23 and 26-48* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,3,4,12,18,22,23 and 26-48* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *513/08; 5/30/08*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. Claims 2, 5-1113-17, 19-21 and 24-25 have been cancelled.

Claims 1, 3-4, 12, 18 and 22 have been amended.

Claims 26-48 have been added.

2. Claims 1, 3-4, 12, 18, 22-23 and 26-48 are pending and under consideration to the extent that the spoondyloarthropathy is psoriatic arthritis, i.e., applicants' elected species.

3. This Office Action contains New Grounds of Rejections

Information Disclosure Statement

5. The information disclosure statement (IDS) submitted on 13 May 2008 and 30 May 2008 have been fully considered by the examiner. A signed and initialed copy of each IDS is included with the instant Office Action.

Objections/Rejections Withdrawn

6. The objection to the specification in the use of various trademarks is withdrawn in view of the amendments to the specification.

7. The objection to claim 15 as depending from withdrawn claim 13 is withdrawn in view of the cancellation of the claim.

8. The rejection of claims 4, 15, 18, 20 and 22-23 under 35 U.S.C. 112, second paragraph, as being indefinite in the recitation of "D2E7" as the sole means of identifying the antibody referred to in the claims is withdrawn in view of the amendments to the claims and the cancellation of claims 15 and 20.

9. The rejection of claims 4 and 15 under 35 U.S.C. 112, second paragraph, as being indefinite in the recitation "antigen-binding fragment thereof, is D2E7" is withdrawn in view of the amendments to claim 4 and the cancellation of claim 15.

10. The rejection of claims 4, 15, 18, 20 and 22-23 under 35 U.S.C. § 112, first paragraph, because the specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention, because the specification does not provide evidence that the claimed biological materials are (1) known and readily available to the public; (2) reproducible from the written description is withdrawn in view of applicants' arguments, i.e., D2E7 is also known as HUMIRA® and adalimumab and is readily available to the public or commercially available.

11. The rejection of claims 2 and 6 under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of treating psoriatic arthritis in a subject comprising administering a human anti-human TNFα antibody or antigen-binding fragment thereof comprising a light chain comprising CDR1 of SEQ ID NO:7, CDR2 of SEQ ID NO:5 and CDR3 of SEQ ID NO:3, optionally comprising the recited amino acid substitutions and a heavy chain comprising CDR1 of SEQ ID NO:8, CDR2 of SEQ ID NO:6 and CDR3 of SEQ ID NO:4, optionally comprising the recited amino acid substitutions, does not reasonably provide enablement for a method of treating psoriatic arthritis in a subject comprising administering a human anti-human TNFα antibody or antigen-binding fragment thereof comprising a light chain comprising CDR3 of SEQ ID NO:3, optionally comprising the recited amino acid substitutions and a heavy chain comprising a heavy chain comprising CDR3 of SEQ ID NO:4, optionally comprising the recited amino acid substitutions as broadly encompassed by the claims is withdrawn in view of the cancellation of the claims.

12. The rejection of claims 1-4, 6, 12, 14-15, 18, 20 and 22-23 under 35 U.S.C. 103(a) as being unpatentable over Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001) in view of Salfeld et al [a] (WO 97/29131, publication date 8/14/1997, IDS reference A4 filed 4/6/04) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) is withdrawn in view of the amendments to the claims and the cancellation of claims 2, 6, 14-15 and 20.

13. The rejection of claims 1-4, 6, 12, 14-15, 18, 20 and 22-23 are rejected under 35 U.S.C. 103(a) as being unpatentable over Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001) in view of Salfeld et al [b] (U.S. Patent 6,509,015 B1, 2/9/1996, IDS reference A2 filed 4/6/04) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) is withdrawn in view of the amendments to the claims and the cancellation of claims 2, 6, 14-15 and 20.

14. The rejection of claims 1-4, 6, 12, 14-15, 18, 20 and 22-23 on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1-7, 36-39 and 69 of U.S. Patent No. 6,509,015 B1 in view of Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) is withdrawn in view of the amendments to the claims and the cancellation of claims 2, 6, 14-15 and 20.

15. The provisional rejection of claims 1-4, 6, 12, 14-15, 18, 20 and 22-23 on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 2, 11-15, 22-23, 73-77, 80, 82-83, 89-94, 96 and 99 of copending Application No. 10/163,657 in view of Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001) and Salfeld et al [a] (WO 97/29131, publication date 8/14/1997, IDS reference A4 filed 4/6/04) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) is withdrawn in view of the cancellation of claims 2, 11-15, 22-23, 73-77, 80, 82-83, 89-94, 96 and 99 of copending Application No. 10/163,657.

16. The provisional rejection of claims 1-4, 6, 12, 14-15, 18, 20 and 22-23 on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1, 4-8 and 10-14 of copending Application No. 11/435,844 in view of Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) is withdrawn in view of the amendments to the claims.

17. The provisional rejection of claims 1-4, 6, 12, 14-15, 18, 20 and 22-23 on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 15, 19, 56, 66, 77 and 87 of copending Application No.

11/233,252 in view of Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001) and Salfeld et al [a] (WO 97/29131, publication date 8/14/1997, IDS reference A4 filed 4/6/04) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) is withdrawn in view of the amendments to the claims.

18. The provisional rejection of claims 1-4, 6, 12, 14-15, 18, 20 and 22-23 on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1-2 and 4-14 of copending Application No. 10/622,932 in view of Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) is withdrawn in view of the amendments to the claims and in view that the claims of copending Application No. 10/622,932 are drawn to treating psoriasis.

19. The provisional rejection of claims 1-4, 6, 12, 14-15, 18, 20 and 22-23 on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1-11 of copending Application No. 10/623,075 in view of Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) is withdrawn in view of the abandonment of copending Application No. 10/623,075.

20. The provisional rejection of claims 1-4, 6, 12, 14-15, 18, 20 and 22-23 on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1, 3-14 and 16 of copending Application No. 10/623,318 in view of Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) withdrawn in view of the amendments to the claims and in view that the claims of copending Application No. 10/623,318 are drawn to treating juvenile rheumatoid arthritis.

Objections/Rejections Maintained

21. The objection to the specification as disclosing various non-provisional US Application numbers whose status has changed and require updating is maintained.

Applicant's amendment filed 3/24/2008 updating the status of the disclosed non-provisional applications is acknowledged, however, in view that USSNs 10/163,657 and 10/622,932 are pending and may require updating during the pendency of the instant application, the objection is being maintained for convenience.

22. The objection to the title of the invention as not descriptive or clearly indicative of the invention to which the claims are directed is maintained.

The response filed 3/24/2008 does not address the objection and as such the objection is maintained. Applicant should restrict the title to the treatment of psoriatic arthritis using human TNFα antibodies.

23. The provisional rejection of claims 1-4, 6, 12, 14-15, 18, 20 and 22-23 are on the ground of nonstatutory obviousness-type double patenting as being unpatentable over 1, 3-10, 16-21, 78-79, 81, 84, 86-88, 95, 97-98 and newly added claims 100-104 of copending Application No. 10/163,657 in view of Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001) and Salfeld et al [a] (WO 97/29131, publication date 8/14/1997, IDS reference A4 filed 4/6/04) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) is maintained.

The response filed 3/24/2008 states that the rejection is provisional in nature and will be addressed when appropriate, i.e., when the nonstatutory obviousness-type double patenting rejection is the only rejection remaining in the later-filed application. Applicants' remarks are acknowledged, however, in view that the claims are rejected on other grounds and not presently in condition for allowance, the rejection is maintained.

Applicant is reminded that the U.S. Patent and Trademark Office normally will not institute an interference between applications or a patent and an application of common ownership (see MPEP Chapter 2300). Commonly assigned copending Application No. 10/163,657, discussed above, would form

the basis for a rejection of the noted claims under 35 U.S.C. 103(a) if the commonly assigned case qualifies as prior art under 35 U.S.C. 102(e), (f) or (g) and the conflicting inventions were not commonly owned at the time the invention in this application was made. In order for the examiner to resolve this issue, the assignee can, under 35 U.S.C. 103(c) and 37 CFR 1.78(c), either show that the conflicting inventions were commonly owned at the time the invention in this application was made, or name the prior inventor of the conflicting subject matter.

A showing that the inventions were commonly owned at the time the invention in this application was made will preclude a rejection under 35 U.S.C. 103(a) based upon the commonly assigned case as a reference under 35 U.S.C. 102(f) or (g), or 35 U.S.C. 102(e) for applications pending on or after December 10, 2004.

New Grounds of Rejections

Claim Rejections - 35 USC § 103

24. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

The factual inquiries set forth in *Graham* **v.** *John Deere Co.*, 383 U.S. 1, 148 USPQ 459 (1966), that are applied for establishing a background for determining obviousness under 35 U.S.C. 103(a) are summarized as follows:

1. Determining the scope and contents of the prior art.
2. Ascertaining the differences between the prior art and the claims at issue.
3. Resolving the level of ordinary skill in the pertinent art.
4. Considering objective evidence present in the application indicating obviousness or nonobviousness.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

25. Claims 1, 3-4, 12, 18, 22-23 and 26-48 are rejected under 35 U.S.C. 103(a) as being unpatentable over Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001, cited on PTO-892 mailed 9/24/07) in view of Salfeld et al [a] (WO 97/29131, publication date 8/14/1997, IDS reference A4 filed 4/6/04) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980, cited on PTO-892 mailed 9/24/07) and Keystone et al ("The Fully Human Anti-TNF Monoclonal Antibody, Adalimumab (D2E7), Dose Ranging Study: The 24-Week Clinical Results in Patients with Active RA on Methotrexate Therapy (The ARMADA Trial)", *Presented at the Annual Meeting of the Against Rheumatoid Arthritis (EULAR), Prague, Czech Republic,* 2001, IDS reference C64 filed 5/13/08).

The claims are drawn to a method of treating psoriasis in a subject comprising biweekly, subcutaneous administration of a unit dosage form comprising 10-150 mg, 20-80 mg or about 40 mg of an anti-TNFα antibody or antigen-binding fragment thereof such that psoriasis is treated, wherein the anti-TNFα antibody or antigen-binding fragment thereof dissociates from human TNFα with a K_d of 1×10^{-8} M or less and a K_{off} of 1×10^{-3} s^{-1} or less, as determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1×10^{-7} M or less, or comprises a light chain variable region comprising SEQ ID NO:1 and a heavy chain variable region comprising SEQ ID NO:2 or the antibody is D2E7, or is

administered with one additional therapeutic agent selected from the group consisting of ibuprofen, diclofenac and misoprostol, naproxen, meloxicam, indomethacin and diclofenac.

Ogilvie et al teach a method of treating psoriatic arthritis in patients comprising administering the chimeric anti-TNFα monoclonal antibody, Infliximab, which resulted in a dramatic improvement in arthritis and showed rapid improvement of their psoriatic arthritis (see entire document, particularly Fig. 1). Ogilvie et al do not biweekly, subcutaneous administration of a unit dosage form comprising 10-150 mg, 20-80 mg or about 40 mg of an anti-TNFα antibody or antigen-binding fragment thereof such that psoriasis is treated, wherein the anti-TNFα antibody or antigen-binding fragment thereof dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and a K_{off} of 1 x 10^{-3} s^{-1} or less, as determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less, or comprises a light chain variable region comprising SEQ ID NO:1 and a heavy chain variable region comprising SEQ ID NO:2 or the antibody is D2E7, or administration with one additional therapeutic agent selected from the group consisting of ibuprofen, diclofenac and misoprostol, naproxen, meloxicam, indomethacin and diclofenac. These deficiencies are made up for in the teachings of Salfeld et al [a] and Smith et al and Keystone et al.

Salfeld et al [a] teach that because humanized antibodies still retain some of murine sequence, they still may elicit an unwanted immune reaction in human patients and Salfeld et al [a] teach a method for treating TNFα-related disorders in a subject comprising administering a therapeutically effective amount of the neutralizing, high affinity fully human D2E7 anti-human TNFα antibody or antigen-binding fragment thereof identical to the claimed human anti-human TNFα antibodies, i.e., dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and has a K_{off} of 1 x 10^{-3} s^{-1} or less, as determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less, and wherein the human antibody or

antigen-binding fragment thereof comprises a light chain variable region comprising SEQ ID NO:1 and a heavy chain variable region comprising SEQ ID NO:2 and is administered with one or more additional therapeutic agents, including corticosteroids (see entire document, particularly pp. 2-4, 5-6, 12-15, 29-31 and 35-40). Salfeld also teaches a variety of administration regimens, routes of administration, antibody fragments, antibody heavy chain constant regions, and dosages, such as 0.1-20 mg/kg (see entire document, in particular pp. 33-34). Salfeld also teaches that "[d]osage regimens may be adjusted to provide the optimum desired response (e.g., a therapeutic or prophylactic response). For example, a single bolus may be administered, several divided doses may be administered over time or the dose may be proportionally reduced or increased as indicated by the exigencies of the therapeutic situation… It is to be further understood that for any particular subject, specific dosage regimens should be adjusted over time according to the individual need and the professional judgment of the person administering or supervising the administration of the compositions, and that dosage ranges set forth herein are exemplary only and are not intended to limit the scope or practice of the claimed composition (see pp. 33-34). Thus, according to the teaching of Salfeld, *the dosage regimen for anti-TNFα antibody, including dosage scheduling and amount, is a recognized results-effective variable,* i.e., a variable that is recognized as important for therapeutic use of an anti-TNFα antibody and which therefore can be optimized by routine experimentation. See M.P.E.P. § 2144.05 II.B. and *In re Antonie*, 559 F.2d 618, 195 USPQ 6 (CCPA 1977).

Smith et al teach that administration of ibuprofen in patients suffering from psoriatic arthritis effectively decreases pain and joint swelling (see entire document).

Keystone et al teach that the fully human anti- TNFα antibody D2E7 administered subcutaneously at 20 mg, 40 mg and 80 mg every other week (i.e., biweekly) was well tolerated and therapeutically effective, particularly at 40 mg every other week (see entire document).

It would have been *prima facie* obvious to one of ordinary skill in the art at the time the claimed invention was made to use the neutralizing, high affinity D2E7 human anti-human TNFα antibody or an antigen-binding fragment thereof of Salfeld et al [a], administered subcutaneously every other week at 20 mg, 40 mg or 80 mg (particularly 40 mg) with ibuprofen for therapeutic benefit in psoriatic arthritis patients as taught by Oh et al.

One of ordinary skill in the art would have been motivated to and had a reasonable expectation of success at the time the invention was made to use the neutralizing, high affinity D2E7 human anti-human TNFα antibody or an antigen-binding fragment thereof of Salfeld et al [a], administered subcutaneously every other week at 20 mg, 40 mg or 80 mg (particularly 40 mg) with ibuprofen for therapeutic benefit in psoriatic arthritis patients in view of Ogilvie et al and Smith and Keystone et al et al because Ogilvie et al teach a method of treating psoriatic arthritis in patients comprising administering the chimeric anti-TNFα monoclonal antibody, Infliximab, which resulted in a dramatic improvement in arthritis and showed rapid improvement of their psoriatic arthritis, however, Salfeld et al [a] teach that because chimeric and humanized antibodies still retain some of murine sequence, they still may elicit an unwanted immune reaction in human patients and Salfeld et al [a] teach the neutralizing, high affinity D2E7 human anti-human TNFα antibody and antigen-binding fragments thereof for treating TNFα-related disorders in a subject comprising administering a therapeutically effective amount of a human anti-human TNFα antibody or antigen-binding fragment thereof identical to the claimed human anti-human TNFα antibodies, i.e., identical structures/sequences, binding kinetics and neutralization properties (discussed supra) and administered with one or more additional therapeutic agents and Smith et al teach that administration of ibuprofen in psoriatic arthritis patients effectively decreases pain and joint swelling and Keystone et al teach that the fully human anti- TNFα antibody D2E7 administered subcutaneously at 20 mg, 40 mg and 80 mg every other week (i.e., biweekly) was well tolerated and therapeutically effective, particularly at 40 mg every other week. Therefore, one

of ordinary skill in the art would have been motivated to modify the method of Ogilvie et al and administer the D2E7 human anti-human TNFα antibody and antigen-binding fragments thereof of Salfeld et al [a] in combination with ibuprofen in order to avoid any unwanted immune reaction in human patients due to the presence of murine sequences in the chimeric anti-TNFα antibody of Ogilvie et al and reduce pain and joint swelling in psoriatic arthritis patients. Additionally, one of ordinary skill in the art would have been motivated to administer the D2E7 antibody or antigen-binding fragments thereof subcutaneously at 20 mg, 40 mg or 80 mg every other week, which is well tolerated and therapeutically effective according to Keystone et al. The strongest rationale for combining references is a recognition, expressly or impliedly in the prior art or drawn from a convincing line of reasoning based on established scientific principles or legal precedent, that some advantage or expected beneficial result would have been produced by their combination. *In re Sernaker*, 702 F.2d 989, 994-95, 217 USPQ 1, 5-6 (Fed. Cir. 1983). See also, *KSR International Co. v. Teleflex Inc.*, 82 USPQ2d 1385 (U.S. 2007). Thus, it would have been *prima facie* obvious to one skilled in the art at the time the invention was made to produce a method of treating psoriatic arthritis in a human patient comprising biweekly, subcutaneous administration of the neutralizing, high affinity D2E7 human anti-human TNFα antibodies or an antigen-binding fragment thereof at 20 mg, 40 mg or 80 mg and administered with at ibuprofen in view of the teachings of Ogilvie et al and Salfeld et al [a] and Smith et al and Keystone et al.

Therefore, the invention as a whole was *prima facie* obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references.

26. Claims 1, 3-4, 12, 18, 22-23 and 26-48 are rejected under 35 U.S.C. 103(a) as being unpatentable over Ogilvie et al (British Journal of Dermatology,

144(3):587-589, March 2001) in view of Salfeld et al [b] (U.S. Patent 6,509,015 B1, 2/9/1996, IDS reference A2 filed 4/6/04) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) and Keystone et al ("The Fully Human Anti-TNF Monoclonal Antibody, Adalimumab (D2E7), Dose Ranging Study: The 24-Week Clinical Results in Patients with Active RA on Methotrexate Therapy (The ARMADA Trial)", *Presented at the Annual Meeting of the Against Rheumatoid Arthritis (EULAR), Prague, Czech Republic,* 2001, IDS reference C64 filed 5/13/08).

The applied reference has a common inventor with the instant application. Based upon the earlier effective U.S. filing date of the reference, it constitutes prior art under 35 U.S.C. 102(e). This rejection under 35 U.S.C. 102(e) might be overcome either by a showing under 37 CFR 1.132 that any invention disclosed but not claimed in the reference was derived from the inventor of this application and is thus not the invention "by another," or by an appropriate showing under 37 CFR 1.131.

The claims and their interpretation have been described supra.

Ogilvie et al have been described supra. Ogilvie et al do not biweekly, subcutaneous administration of a unit dosage form comprising 10-150 mg, 20-80 mg or about 40 mg of an anti-TNFα antibody or antigen-binding fragment thereof such that psoriasis is treated, wherein the anti-TNFα antibody or antigen-binding fragment thereof dissociates from human TNFα with a K_d of 1×10^{-8} M or less and a K_{off} of 1×10^{-3} s^{-1} or less, as determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1×10^{-7} M or less, or comprises a light chain variable region comprising SEQ ID NO:1 and a heavy chain variable region comprising SEQ ID NO:2 or the antibody is D2E7, or administration with one additional therapeutic agent selected from the group consisting of ibuprofen, diclofenac and misoprostol, naproxen, meloxicam, indomethacin and diclofenac. These deficiencies are made up for in the teachings of Salfeld et al [b] and Smith et al and Keystone et al.

Salfeld et al [b] teach that because humanized antibodies still retain some of murine sequence, they still may elicit an unwanted immune reaction in human patients and Salfeld et al [b] teach a method for treating TNFα-related disorders in a subject comprising administering a therapeutically effective amount of the neutralizing, high affinity fully human D2E7 anti-human TNFα antibody or antigen-binding fragment thereof identical to the claimed human anti-human TNFα antibodies, i.e., dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and has a K_{off} of 1 x 10^{-3} s^{-1} or less, as determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less, and wherein the human antibody or antigen-binding fragment thereof comprises a light chain variable region comprising SEQ ID NO:1 and a heavy chain variable region comprising SEQ ID NO:2 and is administered with one or more additional therapeutic agents, including corticosteroids (see entire document, particularly pp. 2-4, 5-6, 12-15, 29-31 and 35-40). Salfeld [b] also teaches a variety of administration regimens, routes of administration, antibody fragments, antibody heavy chain constant regions, and dosages, such as 0.1-20 mg/kg (see entire document, in particular pp. 33-34). Salfeld [b] also teaches that "[d]osage regimens may be adjusted to provide the optimum desired response (e.g., a therapeutic or prophylactic response). For example, a single bolus may be administered, several divided doses may be administered over time or the dose may be proportionally reduced or increased as indicated by the exigencies of the therapeutic situation… It is to be further understood that for any particular subject, specific dosage regimens should be adjusted over time according to the individual need and the professional judgment of the person administering or supervising the administration of the compositions, and that dosage ranges set forth herein are exemplary only and are not intended to limit the scope or practice of the claimed composition (see pp. 33-34). Thus, according to the teaching of Salfeld [b], *the dosage regimen for anti-TNFα antibody, including dosage scheduling and amount, is a recognized results-effective variable,* i.e., a variable that is

recognized as important for therapeutic use of an anti-TNFα antibody and which therefore can be optimized by routine experimentation. See M.P.E.P. § 2144.05 II.B. and *In re Antonie*, 559 F.2d 618, 195 USPQ 6 (CCPA 1977).

Smith et al have been described supra.

Keystone et al have been described supra.

It would have been *prima facie* obvious to one of ordinary skill in the art at the time the claimed invention was made to use the neutralizing, high affinity D2E7 human anti-human TNFα antibody or an antigen-binding fragment thereof of Salfeld et al [b], administered subcutaneously every other week at 20 mg, 40 mg or 80 mg (particularly 40 mg) with ibuprofen for therapeutic benefit in psoriatic arthritis patients as taught by Ogilvie et al.

One of ordinary skill in the art would have been motivated to and had a reasonable expectation of success at the time the invention was made to use the neutralizing, high affinity D2E7 human anti-human TNFα antibody or an antigen-binding fragment thereof of Salfeld et al [b], administered subcutaneously every other week at 20 mg, 40 mg or 80 mg (particularly 40 mg) with ibuprofen for therapeutic benefit in psoriatic arthritis patients in view of Ogilvie et al and Smith and Keystone et al et al because Ogilvie et al teach a method of treating psoriatic arthritis in patients comprising administering the chimeric anti-TNFα monoclonal antibody, Infliximab, which resulted in a dramatic improvement in arthritis and showed rapid improvement of their psoriatic arthritis, however, Salfeld et al [b] teach that because chimeric and humanized antibodies still retain some of murine sequence, they still may elicit an unwanted immune reaction in human patients and Salfeld et al [b] teach the neutralizing, high affinity D2E7 human anti-human TNFα antibody and antigen-binding fragments thereof for treating TNFα-related disorders in a subject comprising administering a therapeutically effective amount of a human anti-human TNFα antibody or antigen-binding fragment thereof identical to the claimed human anti-human TNFα antibodies, i.e., identical structures/sequences, binding kinetics and neutralization properties (discussed supra) and administered with one or more additional therapeutic

agents and Smith et al teach that administration of ibuprofen in psoriatic arthritis patients effectively decreases pain and joint swelling and Keystone et al teach that the fully human anti- TNFα antibody D2E7 administered subcutaneously at 20 mg, 40 mg and 80 mg every other week (i.e., biweekly) was well tolerated and therapeutically effective, particularly at 40 mg every other week. Therefore, one of ordinary skill in the art would have been motivated to modify the method of Ogilvie et al and administer the D2E7 human anti-human TNFα antibody and antigen-binding fragments thereof of Salfeld et al [b] in combination with ibuprofen in order to avoid any unwanted immune reaction in human patients due to the presence of murine sequences in the chimeric anti-TNFα antibody of Ogilvie et al and reduce pain and joint swelling in psoriatic arthritis patients. Additionally, one of ordinary skill in the art would have been motivated to administer the D2E7 antibody or antigen-binding fragments thereof subcutaneously at 20 mg, 40 mg or 80 mg every other week, which is well tolerated and therapeutically effective according to Keystone et al. The strongest rationale for combining references is a recognition, expressly or impliedly in the prior art or drawn from a convincing line of reasoning based on established scientific principles or legal precedent, that some advantage or expected beneficial result would have been produced by their combination. *In re Sernaker*, 702 F.2d 989, 994-95, 217 USPQ 1, 5-6 (Fed. Cir. 1983). See also, *KSR International Co. v. Teleflex Inc.*, 82 USPQ2d 1385 (U.S. 2007). Thus, it would have been *prima facie* obvious to one skilled in the art at the time the invention was made to produce a method of treating psoriatic arthritis in a human patient comprising biweekly, subcutaneous administration of the neutralizing, high affinity D2E7 human anti-human TNFα antibody or an antigen-binding fragment thereof at 20 mg, 40 mg or 80 mg and administered with at ibuprofen in view of the teachings of Ogilvie et al and Salfeld et al [b] and Smith et al and Keystone et al.

Therefore, the invention as a whole was *prima facie* obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references.

Double Patenting

27. The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

28. Claims 1, 3-4, 12, 18, 22-23 and 26-48 are rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1-7, 36-39 and 69 of U.S. Patent No. 6,509,015 B1 in view of Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) and Keystone et al ("The Fully Human Anti-TNF Monoclonal Antibody, Adalimumab (D2E7), Dose Ranging Study: The 24-Week Clinical Results in Patients with Active RA on Methotrexate Therapy (The ARMADA Trial)", *Presented at the Annual Meeting of the Against*

Rheumatoid Arthritis (EULAR), Prague, Czech Republic, 2001, IDS reference C64 filed 5/13/08). Although the conflicting claims are not identical, they are not patentably distinct from each other.

The instant claims have been described supra.

Claims 1-7, 36-39 and 69-70 of U.S. Patent No. 6,509,015 B1 are drawn to a method of inhibiting human TNFα activity in a human subject and a method of treating a human subject suffering from a disorder in which TNFα activity is detrimental comprising administering to the human subject a human anti-human TNFα antibody or antigen-binding fragment thereof that is identical to the neutralizing, high affinity human anti-human TNFα antibodies and antigen-binding fragments thereof of the present claims, i.e., identical structures/sequences, binding kinetics and neutralization properties and wherein the administered human anti-human TNFα antibody or antigen binding fragment thereof is optionally administered with at least one additional therapeutic agent. Claims 1-7, 36-39 and 69-70 of U.S. Patent No. 6,509,015 B1 do not teach biweekly, subcutaneous administration of a unit dosage form comprising 10-150 mg, 20-80 mg or about 40 mg of the human anti-human TNFα antibody or antigen-binding fragment thereof for the treatment of psoriasis in a human patient or wherein the additional therapeutic agent is ibuprofen. These deficiencies are made up for in the teachings of Ogilvie et al and Smith et al and Keystone et al.

Ogilvie et al have been described supra.

Smith et al have been described supra.

Keystone et al have been described supra.

The claims in the instant application are obvious variants of claims 1-7, 36-39 and 69 of U.S. Patent No. 6,509,015 B1 because it would have been *prima facie* obvious to one of ordinary skill in the art at the time the claimed invention was made to treat psoriatic arthritis in a human patient comprising biweekly, subcutaneous administration at a dose of 20 mg, 40 mg or 80 mg of the human anti-human TNFα antibodies or antigen-binding fragments thereof of claims 1-7, 36-39 and 69-70 of U.S. Patent No. 6,509,015 B1 and administered

with ibuprofen according in view of the teachings of Ogilvie et al and Smith et al and Keystone et al.

One of ordinary skill in the art would have been motivated to and had a reasonable expectation of success at the time the invention was made to treat psoriatic arthritis in a human patient comprising biweekly, subcutaneous administration at a dose of 20 mg, 40 mg or 80 mg of the human anti-human TNFα antibodies or antigen-binding fragments thereof of claims 1-7, 36-39 and 69-70 of U.S. Patent No. 6,509,015 B1 and administered with ibuprofen according in view of the teachings of Ogilvie et al and Smith et al and Keystone et al because Ogilvie et al teach a method of treating psoriatic arthritis in patients comprising administering the anti-TNFα monoclonal antibody, Infliximab, which resulted in a dramatic improvement in arthritis and showed rapid improvement of their psoriatic arthritis and Smith et al teach that administration of ibuprofen in psoriatic arthritis patients effectively decreases pain and joint swelling and Keystone et al teach that the fully human anti- TNFα antibody D2E7 administered subcutaneously at 20 mg, 40 mg and 80 mg every other week (i.e., biweekly) was well tolerated and therapeutically effective, particularly at 40 mg every other week. Thus, it would have been *prima facie* obvious to one of ordinary skill in the art at the time the claimed invention was made to treat psoriatic arthritis in a human patient comprising biweekly, subcutaneous administration at a dose of 20 mg, 40 mg or 80 mg of the human anti-human TNFα antibodies or antigen-binding fragments thereof of claims 1-7, 36-39 and 69-70 of U.S. Patent No. 6,509,015 B1 and administered with ibuprofen according in view of the teachings of Ogilvie et al and Smith et al and Keystone et al.

Claims 1, 3-4, 12, 18, 22-23 and 26-48 are directed to an invention not patentably distinct from claim 1-7, 36-39 and 69 of commonly assigned U.S. Patent No. 6,509,015 B1. Specifically, see above.

The U.S. Patent and Trademark Office normally will not institute an interference between applications or a patent and an application of common ownership (see MPEP Chapter 2300). Commonly assigned U.S. Patent No.

6,509,015 B1, discussed above, would form the basis for a rejection of the noted claims under 35 U.S.C. 103(a) if the commonly assigned case qualifies as prior art under 35 U.S.C. 102(e), (f) or (g) and the conflicting inventions were not commonly owned at the time the invention in this application was made. In order for the examiner to resolve this issue, the assignee can, under 35 U.S.C. 103(c) and 37 CFR 1.78(c), either show that the conflicting inventions were commonly owned at the time the invention in this application was made, or name the prior inventor of the conflicting subject matter.

A showing that the inventions were commonly owned at the time the invention in this application was made will preclude a rejection under 35 U.S.C. 103(a) based upon the commonly assigned case as a reference under 35 U.S.C. 102(f) or (g), or 35 U.S.C. 102(e) for applications pending on or after December 10, 2004.

29. Claims 1, 3-4, 12, 18, 22-23 and 26-48 are provisionally rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1, 4-5, 8-11, 14, 38-39, 49-50, 52-53 and 55-57 of copending Application No. 11/435,844 in view of Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) and Keystone et al ("The Fully Human Anti-TNF Monoclonal Antibody, Adalimumab (D2E7), Dose Ranging Study: The 24-Week Clinical Results in Patients with Active RA on Methotrexate Therapy (The ARMADA Trial)", *Presented at the Annual Meeting of the Against Rheumatoid Arthritis (EULAR), Prague, Czech Republic,* 2001, IDS reference C64 filed 5/13/08). Although the conflicting claims are not identical, they are not patentably distinct from each other.

The instant claims have been described supra.
Claims 1, 4-5, 8-11, 14, 38-39, 49-50, 52-53 and 55-57 of copending Application No. 11/435,844 are drawn to a method for treating a human subject suffering

from erosive polyarthritis, or psoriatic arthritis comprising administering to the subject a TNFα antibody or antigen-binding fragment thereof such that erosive polyarthritis is treated, wherein the antibody or antigen-binding fragment thereof is human, dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and has a K_{off} of 1 x 10^{-3} s^{-1} or less, as determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less, and wherein the human antibody or antigen-binding fragment thereof comprises a light chain variable region comprising SEQ ID NO:1 and a heavy chain variable region comprising SEQ ID NO:2, all properties of the human anti-human TNFα antibodies and antigen-binding fragments thereof of the present claims and wherein the TNFα antibody or antigen-binding fragment thereof is administered on a biweekly dosing regimen which is about 40 mg and administered with a therapeutic agent. Claims 1, 4-5, 8-11, 14, 38-39, 49-50, 52-53 and 55-57 of copending Application No. 11/435,844 do not teach biweekly, subcutaneous administration of a unit dosage form comprising 10-150 mg, 20-80 mg or about 40 mg of the human anti-human TNFα antibody or antigen-binding fragment thereof for the treatment of psoriasis in a human patient or wherein the additional therapeutic agent is ibuprofen. These deficiencies are made up for in the teachings of Ogilvie et al and Smith et al and Keystone et al.

Ogilvie et al have been described supra.

Smith et al have been described supra.

The claims in the instant application are obvious variants of claims 1, 4-5, 8-11, 14, 38-39, 49-50, 52-53 and 55-57 of copending Application No. 11/435,844 because it would have been *prima facie* obvious to one of ordinary skill in the art at the time the claimed invention was made to treat psoriatic arthritis in a human patient comprising biweekly, subcutaneous administration at a dose of 20 mg, 40 mg or 80 mg of the human anti-human TNFα antibodies or antigen-binding fragments thereof of claims 1, 4-5, 8-11, 14, 38-39, 49-50, 52-53 and 55-57 of copending Application No. 11/435,844 and administered with

ibuprofen according in view of the teachings of Ogilvie et al and Smith et al and Keystone et al.

One of ordinary skill in the art would have been motivated to and had a reasonable expectation of success at the time the invention was made to treat psoriatic arthritis in a human patient comprising biweekly, subcutaneous administration at a dose of 20 mg, 40 mg or 80 mg of the human anti-human TNFα antibodies or antigen-binding fragments thereof of claims 1, 4-5, 8-11, 14, 38-39, 49-50, 52-53 and 55-57 of copending Application No. 11/435,844 and administered with ibuprofen according in view of the teachings of Ogilvie et al and Smith et al and Keystone et al because Ogilvie et al teach a method of treating psoriatic arthritis in patients comprising administering the anti-TNFα monoclonal antibody, Infliximab, which resulted in a dramatic improvement in arthritis and showed rapid improvement of their psoriatic arthritis and Smith et al teach that administration of ibuprofen in psoriatic arthritis patients effectively decreases pain and joint swelling and Keystone et al teach that the fully human anti- TNFα antibody D2E7 administered subcutaneously at 20 mg, 40 mg and 80 mg every other week (i.e., biweekly) was well tolerated and therapeutically effective, particularly at 40 mg every other week. Thus, it would have been *prima facie* obvious to one of ordinary skill in the art at the time the claimed invention was made to treat psoriatic arthritis in a human patient comprising biweekly, subcutaneous administration at a dose of 20 mg, 40 mg or 80 mg of the human anti-human TNFα antibodies or antigen-binding fragments thereof of claims 1, 4-5, 8-11, 14, 38-39, 49-50, 52-53 and 55-57 of copending Application No. 11/435,844 and administered with ibuprofen according in view of the teachings of Ogilvie et al and Smith et al and Keystone et al.

Claims 1, 3-4, 12, 18, 22-23 and 26-48 are directed to an invention not patentably distinct from claim 1, 4-5, 8-11, 14, 38-39, 49-50, 52-53 and 55-57 of commonly assigned copending Application No. 11/435,844. Specifically, see above.

The U.S. Patent and Trademark Office normally will not institute an interference between applications or a patent and an application of common ownership (see MPEP Chapter 2300). Commonly assigned copending Application No. 11/435,844, discussed above, would form the basis for a rejection of the noted claims under 35 U.S.C. 103(a) if the commonly assigned case qualifies as prior art under 35 U.S.C. 102(e), (f) or (g) and the conflicting inventions were not commonly owned at the time the invention in this application was made. In order for the examiner to resolve this issue, the assignee can, under 35 U.S.C. 103(c) and 37 CFR 1.78(c), either show that the conflicting inventions were commonly owned at the time the invention in this application was made, or name the prior inventor of the conflicting subject matter.

A showing that the inventions were commonly owned at the time the invention in this application was made will preclude a rejection under 35 U.S.C. 103(a) based upon the commonly assigned case as a reference under 35 U.S.C. 102(f) or (g), or 35 U.S.C. 102(e) for applications pending on or after December 10, 2004.

This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

30. Claims 1, 3-4, 12, 18, 22-23 and 26-48 are provisionally rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 15, 19, 56, 66, 77 and 87 of copending Application No. 11/233,252 in view of Ogilvie et al (British Journal of Dermatology, 144(3):587-589, March 2001) and Salfeld et al [a] (WO 97/29131, publication date 8/14/1997, IDS reference A4 filed 4/6/04) and Smith et al (Arthritis Rheum. 23(8):961-962, August 1980) and Keystone et al ("The Fully Human Anti-TNF Monoclonal Antibody, Adalimumab (D2E7), Dose Ranging Study: The 24-Week Clinical Results in Patients with Active RA on Methotrexate Therapy (The ARMADA Trial)", *Presented at the Annual Meeting of the Against Rheumatoid Arthritis*

(EULAR), Prague, Czech Republic, 2001, IDS reference C64 filed 5/13/08). Although the conflicting claims are not identical, they are not patentably distinct from each other.

The instant claims have been described supra.

Claims 15, 19, 56, 66, 77 and 87 of copending Application No. 11/233,252 are drawn to a method for treating a subject suffering from a disorder in which TNFα activity is detrimental comprising administering a pharmaceutical composition comprising an isolated human anti-human TNFα antibody or antigen-binding fragment thereof that dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and has a K_{off} of 1 x 10^{-3} s^{-1} or less, as determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less and wherein the pharmaceutical composition is administered with at least one additional therapeutic agent. Claims 15, 19, 56, 66, 77 and 87 of copending Application No. 11/233,252 do not teach biweekly, subcutaneous administration of a unit dosage form comprising 10-150 mg, 20-80 mg or about 40 mg of the human anti-human TNFα antibody or antigen-binding fragment thereof for the treatment of psoriasis in a human patient or wherein the additional therapeutic agent is ibuprofen. These deficiencies are made up for in the teachings of Ogilvie et al and Smith et al and Keystone et al.

Ogilvie et al have been described supra.

Smith et al have been described supra.

Keystone et al have been described supra.

The claims in the instant application are obvious variants of claims 15, 19, 56, 66, 77 and 87 of copending Application No. 11/233,252 because it would have been *prima facie* obvious to one of ordinary skill in the art at the time the claimed invention was made to treat psoriatic arthritis in a human patient comprising biweekly, subcutaneous administration at a dose of 20 mg, 40 mg or 80 mg of the human anti-human TNFα antibodies or antigen-binding fragments thereof of claims 15, 19, 56, 66, 77 and 87 of copending Application No.

11/233,252 and administered with ibuprofen according in view of the teachings of Ogilvie et al and Smith et al and Keystone et al.

One of ordinary skill in the art would have been motivated to and had a reasonable expectation of success at the time the invention was made to treat psoriatic arthritis in a human patient comprising biweekly, subcutaneous administration at a dose of 20 mg, 40 mg or 80 mg of the human anti-human TNFα antibodies or antigen-binding fragments thereof of claims 15, 19, 56, 66, 77 and 87 of copending Application No. 11/233,252 and administered with ibuprofen according in view of the teachings of Ogilvie et al and Smith et al and Keystone et al because Ogilvie et al teach a method of treating psoriatic arthritis in patients comprising administering the anti-TNFα monoclonal antibody, Infliximab, which resulted in a dramatic improvement in arthritis and showed rapid improvement of their psoriatic arthritis and Smith et al teach that administration of ibuprofen in psoriatic arthritis patients effectively decreases pain and joint swelling and Keystone et al teach that the fully human anti- TNFα antibody D2E7 administered subcutaneously at 20 mg, 40 mg and 80 mg every other week (i.e., biweekly) was well tolerated and therapeutically effective, particularly at 40 mg every other week. Thus, it would have been *prima facie* obvious to one of ordinary skill in the art at the time the claimed invention was made to treat psoriatic arthritis in a human patient comprising biweekly, subcutaneous administration at a dose of 20 mg, 40 mg or 80 mg of the human anti-human TNFα antibodies or antigen-binding fragments thereof of claims 15, 19, 56, 66, 77 and 87 of copending Application No. 11/233,252 and administered with ibuprofen according in view of the teachings of Ogilvie et al and Smith et al and Keystone et al.

Claims 1, 3-4, 12, 18, 22-23 and 26-48 are directed to an invention not patentably distinct from claims 15, 19, 56, 66, 77 and 87 of commonly assigned copending Application No. 11/233,252. Specifically, see above.

The U.S. Patent and Trademark Office normally will not institute an interference between applications or a patent and an application of common ownership (see MPEP Chapter 2300). Commonly assigned copending Application No. 11/233,252, discussed above, would form the basis for a rejection of the noted claims under 35 U.S.C. 103(a) if the commonly assigned case qualifies as prior art under 35 U.S.C. 102(e), (f) or (g) and the conflicting inventions were not commonly owned at the time the invention in this application was made. In order for the examiner to resolve this issue, the assignee can, under 35 U.S.C. 103(c) and 37 CFR 1.78(c), either show that the conflicting inventions were commonly owned at the time the invention in this application was made, or name the prior inventor of the conflicting subject matter. A showing that the inventions were commonly owned at the time the invention in this application was made will preclude a rejection under 35 U.S.C. 103(a) based upon the commonly assigned case as a reference under 35 U.S.C. 102(f) or (g), or 35 U.S.C. 102(e) for applications pending on or after December 10, 2004.

This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

31. No claim is allowed.

32. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be

calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to David J. Blanchard whose telephone number is (571) 272-0827. The examiner can normally be reached at Monday through Friday from 8:00 AM to 6:00 PM, with alternate Fridays off. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Larry Helms, can be reached at (571) 272-0832.

The official fax number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

/David J. Blanchard/
Primary Examiner, A.U. 1643